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THE EFFECTS OF ACUTE EXERCISE AND PSYCHOLOGICAL STRESS ON EPISODIC MEMORY

by

Lauren Koehler

A thesis submitted to the faculty of The University of Mississippi in partial fulfillment of the requirements of the Sally McDonnell Barksdale Honors College.

Oxford

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ABSTRACT

LAUREN KOEHLER: The Effects of Acute Exercise and Psychological Stress on Episodic Memory

(Under the direction of Dr. Paul Loprinzi)

Research has suggested that exercise has an effect on memory function. Studies have also shown that exercise mitigates the effects of stress. The relationship between exercise and stress in regard to memory function is noteworthy. There has been little research on this relationship in young adults. This thesis discusses the effects of acute exercise on the episodic memory of young adults when they are exposed to a psychosocial stressor. The results of the research study show that acute aerobic exercise before memory encoding or consolidation was not associated with memory function after exposure to a stressor. Future research in this area is needed.

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List of Abbreviations

fMRI	functional magnetic resonance imaging
HPA	hypothalamic-pituitary-adrenal
BDNF	brain-derived neurotrophic factor
AMPA	α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
cAMP	cyclic adenine monophosphate
CREB	cAMP response element-binding protein
mTOR	mammalian target of rapamycin
LTP	long-term potentiation
5HT	5-hydroxytryptamine
PRP	plasticity related protein
NMDA	N-methyl-D-aspartate
PAR-Q	physical activity readiness questionnaire

Episodic Memory

There are multiple facets of memory that are controlled by various parts of the brain. Memory is broken into two main categories. Declarative memory is memory that is conveyed by "direct conscious access to information" (Dickerson & Eichenbaum, 2009, p. 86). Nondeclarative memory is conveyed unconsciously "through changes in behavioral or physiological responses" (Dickerson & Eichenbaum, 2009, p. 86). Long-term declarative memory is further subdivided into semantic memory and episodic memory. Semantic memory includes the retrieval of learned facts with unknown knowledge of when and where the facts were learned. Episodic memory comprises the learning, storing, and retrieval of events in everyday life. This type of memory includes where and when something occurred and the recall of any details related to an event (Dickerson & Eichenbaum, 2009).

Further, due to human and animal research studies, we know which parts of the brain are involved in episodic memory. In the late 1800s and early 1900s, researchers found that the brainstem, third ventricle, frontal lobe, and hippocampal formation were all involved in episodic memory function (Dickerson & Eichenbaum, 2009). Further animal research supported this showing that the parahippocampal cortical areas and the hippocampus were involved (Dickerson & Eichenbaum, 2009). The medial temporal lobe "has a fully selective function in memory" which neocortical areas do not (Dickerson & Eichenbaum, 2009, p. 89).

With the advancement in neuroimaging through fMRIs and the study of human memory disorders, researchers were able to discover more about episodic memory processes. Neuroimaging helped show the different parts of the brain that are activated during tasks such as encoding and retrieval of memories in the medial temporal lateral region (Dickerson & Eichenbaum, 2009). Also, the study of memory disorders using neuroimaging has provided additional insights on the neuroanatomy and neuropsychology of human memory function. Amnesia, Alzheimer's disease, hippocampal sclerosis, focal lesions, and epilepsy all involve impairment of the medial temporal lateral region and hippocampal regions which prove their involvement in episodic memory function (Dickerson & Eichenbaum, 2009).

Effects of Stress on Episodic Memory

Developments in research have also shown that exercise before a toxic stressor diminishes its negative effect of the stressor on episodic memory (Loprinzi & Frith, 2018a). Through research using animal studies, the effects of acute and chronic stress on exercise have been analyzed. On the contrary, the positive effects of exercise on memory function when a stressor was used are also evident.

Loprinzi and Frith analyzed eight studies that used a preventive model where exercise occurred before the stressor and they found that the stressors damaged memory function. They also looked at ten studies that used a therapeutic model. In eight of these, the stress procedure hindered memory function as well (Loprinzi & Frith, 2018a). The stressors in these studies were separation from

mothers, listening to loud noises, restraint, competition and social loss, and smelling cat odor. 22 of the 23 studies applied chronic stress. All of them used chronic exercise. The memory tasks used were the Morris water maze, object recognition test, or the inhibitory avoidance task (Loprinzi & Frith, 2018a). Various animal studies show that stress does negatively affect memory function.

Although, acute stress in moderate levels can improve memory of emotional-based information. The memory encoding is improved when the stress occurs before the memory task. The consolidation of the memory trace can also be improved when exercise or the stressor occurs before or soon after the memory task. Acute stress enhances consolidation for important information, but negatively affects non-relevant stimuli encoding. It can also impair the retrieval of stimuli for free and cued recall. Although this is true for moderate levels of acute stress, high levels of stressors can impair memory retrieval when the stressor happens near the time of retrieval of an unrelated memory (Loprinzi & Frith, 2018a).

On the contrary, chronic stress can cause negative effects on memory. It increases HPA axis activity. This can cause cells to die and neurons to lose spines and dendrites to shrink. This may be due to increased cortisol levels in the hippocampus (Loprinzi & Frith, 2018a). Stress also can decrease neurotransmitter levels, BDNF levels, AMPA receptor expression, and mineralocorticoid and glucocorticoid receptor expression. Chronic stress also impairs neurogenesis and may cause apoptosis of progenitor cells, which cause the hippocampus to lose

volume (Loprinzi & Frith, 2018a). These parts of the brain are all essential to memory function, so long-term stress may damage these functions.

Exercise can help alleviate the effects of chronic stress on memory function by stimulating the vagus nerve and muscle afferent nerve fibers that project into the hippocampus. This helps facilitate long-term potentiation, which is thought to be a key mechanism underlying episodic memory function. The increase of hormones due to exercise also increases neurotrophic factors, activates transcriptor factor expression, and increases AMPA translocation. Exercise also helps in the "attenuation of HPA axis activity, suppress oxidative stress, facilitate neurogenesis, and regulate mineralocorticoid and glucocorticoid receptor expression" (Loprinzi & Frith, 2018a, p. 9-10). Neurotransmitter production and cell survival are also increased (Loprinzi & Frith, 2018). Exercise works to oppose the negative effects of stress.

Although there are negative effects of chronic stress on memory function, chronic exercise has been shown to alleviate these effects. Different intensities of exercise, timing of events, and types of stress all are major factors in this research field. There are few human studies on this topic currently, particularly as to the potential effects of acute exercise on attenuating stress-induced memory impairment.

Effects of Exercise on Episodic Memory

Exercise has also been seen to enhance episodic memory. The relationship between cognitive function and exercise may be bidirectional (Loprinzi & Frith, 2018b). Chronic exercise has shown to activate neurogenesis, gliogenesis,

angiogenesis, cerebral circulation, and growth factor production. Loprinzi and Frith found that the possible mechanisms of exercise on memory function include:

> exercise enhancing neuronal excitability, exercise enhancing attentional resource allocation to facilitate memory encoding, exercise upregulating α-amino-3-hydroxy-5-methyl-4-isoxazoleproprionic acid receptor levels, opening N-methyl-D-asparate channels (NMDA), increasing excitatory post-synaptic potentials in the hippocampus, exercise priming neurons to be encoded in the memory trace by increasing cAMP response binding (CREB) transcription, brain -derived neurotropic factor (BDNF) expression from exercise, and exercising enhancing dendritic spine growth (p. 286).

Specifically, BDNF seems to play a major role in exercise and memory through the increase of neuronal survival and intracellular calcium levels, facilitation of transcription factors, and induction of mTOR-mediated mRNA translation in memory consolidation (Loprinzi & Frith, 2018b).

One major pathway that is a mechanism of acute exercise on episodic memory function is the muscle spindle pathway. LTP is increased during exercise because when skeletal muscles contract, muscle spindles are activated. This causes action potentials to be sent to the brain stem. These ultimately get sent to the prefrontal cortex and basolateral amygdala which are involved in memory function (Loprinzi & Frith, 2018b).

In addition, acute exercise also causes the vagus nerve to be activated. It is the “longest cranial nerve in the body, comprised approximately 80% afferent fibers and 20% efferent fibers” (Loprinzi & Frith, 2018b, p. 286). Memory-related neurotransmitter levels of norepinephrine, dopamine, serotonin, and acetylcholine can all be increased due to vagus nerve stimulation (Loprinzi & Frith, 2018b).

Neurotransmitters also play a key role in the relationship between exercise and memory. For example, norepinephrine binds to β-receptors which activates LTP. It is evident that norepinephrine plays a major role in hippocampal LTP. Research has shown that exercise can increase norepinephrine levels. Exercise has also caused dopamine levels to increase, but this has been irregular in research studies. Dopamine effects LTP through its signaling that helps “mediate memory trace formation” (Loprinzi & Frith, 2018b, p. 288). 5HT is another important neurotransmitter involved in memory function. It is involved in LTP in the hippocampus. It may be released specifically during acute stress which ultimately activates gene expression. Research has shown that exercise can increase 5HT levels, which, in turn, may help to increase memory function. Acetylcholine is also an important neurotransmitter involved in mediating memory function. Walking has been shown to present the greatest effects on LTP. Exercise has been shown to increase norepinephrine, dopamine, 5HT, and acetylcholine levels which can improve memory function (Loprinzi & Frith, 2018b).

Additionally, synaptic tagging may also increase memory function. Behavioral tagging is the “conversion of a short-term weak memory to a stable long-term memory” and this is due to how close it is to a strong memory

(Loprinzi & Frith, 2018b, p. 291). Plasticity-related protein activation by the stronger memory can enhance the weaker memory by improving LTP at the synapse. Exercise may create these PRPs (Loprinzi & Frith, 2018b).

Furthermore, associativity may affect the effect of acute exercise on episodic memory function. Through LTP and its related mechanisms, exercise may be able to stabilize the memory trace. Associativity is seen when "the weak input is activated around the same time as the strong input," which also starts LTP in these pathways (Loprinzi & Frith, 2018b, p. 292). The strong input is the exercise which may create a high action potential in addition to the memory trace. The action potential may also start a dendritic spike which aids in synaptic depolarization of the memory stimulus. This prepares NMDA structures to facilitate LTP (Loprinzi & Frith, 2018b).

Another mechanism of the effects of acute exercise in episodic memory may be via cognitive attention. This includes alerting, orienting, and executive control in relation to the stimulus. There are two types of attention, which are bottom-up attention and top-down attention. Bottom-up attention refers to when neurons self-activate after a sensory stimulus. The neurons in this group continue to fire once they receive a "transitory superior threshold stimulus," and are "activated by another source of excitatory input" (Loprinzi & Frith, 2018b, p. 293). Acute exercise has shown to activate neuronal excitation in the mesencephalic reticular formation, thalamus, and limbic structures. Top-down attention is a voluntary, high-order process involving the frontal and parietal structures. These structures merge information from the neurons in the bottom-up

structures. Acute exercise may assist in bottom-up and top-down attention by increasing neuronal activity and excitement within the structures involved (Loprinzi & Frith, 2018b).

Although chronic exercise has helped factors that affect memory function, acute exercise may improve episodic memory function through the muscle spindle and vagus nerve pathways, neurotransmitter mediation, synaptic tagging, associativity, and attention. More research is needed to evaluate the effect of acute exercise on episodic memory function during different exercise intensities and lengths of time. Varying these factors could give more information about the relationship between exercise and episodic memory (Loprinzi & Frith, 2018b).

Purpose of the Study

The purpose of my research study was to evaluate whether exercise is associated with episodic memory function even when exposed to a psychosocial stressor. We wanted to evaluate whether exercise prior to memory encoding or during the memory consolidation process can affect episodic memory function after a stressful event has occurred. Memory function is critical for daily life. Therefore, overall health is significantly affected by the ability to remember. As exercise is another major health factor, its relationship to memory is of great interest as well. There has been little research in young adults on how acute exercise affects episodic memory function when exposed to a psychosocial stressor, so this study sought to analyze this relationship. We aimed to identify if acute exercise before a psychological stressor can improve episodic memory function (Loprinzi et al., 2019).

Methods

Study Design

See Figure 1 for a schematic of the study design. All participants completed a single laboratory visit, involving a between-subject design. Participants were randomized into a control and exercise group with a sample size of 20 participants in each group. The exercise group walked on a treadmill for 15 minutes at a moderate intensity that aimed to elicit a physiological response of 50-59% of their estimated heart rate reserve. The exercise bout was followed by a 5 minute resting period. The control group rested for 20 minutes instead of doing the exercise bout. After completing either the exercise bout or control period, they completed the Trier Social Stress Test. Before and after the test, the participants completed a Feeling Scale and Felt Arousal Scale. The stress test consisted of a 5-minute speech and 5-minute arithmetic task with two researchers present to increase stress levels. The instructions for the social stress test are shown in Table 3. After this, they did a baseline memory task, which was the Logical Memory Test. After resting for 35 minutes, the participants completed the post-memory recall task. The control group followed the same protocol, but did not participate in the exercise bout. Therefore, the temporal time period was the same for both groups. The participants were not informed of the stress test before they participated in the study so that the effects of the stress would not be attenuated from a potential anticipatory effect. They were all informed of the real purpose of the study at the end of the visit, and all reconsented.

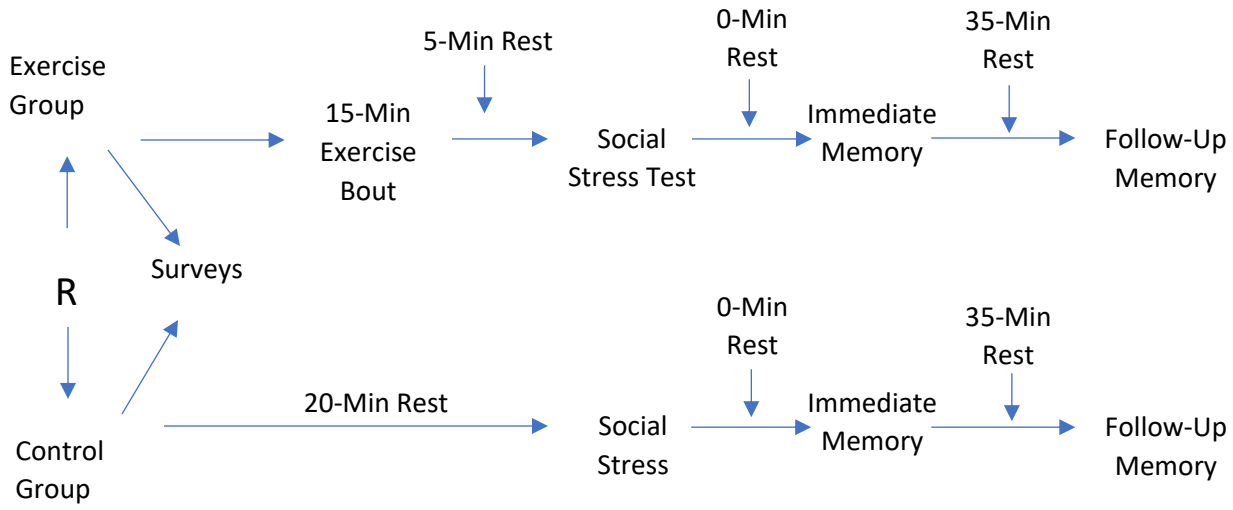

Figure 1. Schematic of the study design.

Participants

The participants consisted of 40 college students because this is similar to other related experiments on this topic. This was based on an a-priori power analysis indicating that 36 total participants would be needed to achieve adequate statistical power (1-β error probability of 0.90), with inputs of 0.05 (α error probability), 2 groups, 2 memory assessments per group, and an estimated partial eta-squared of 0.05 (Loprinzi, Blough, Crawford, et al., 2019). Participants were ineligible for this study if they were outside the 18-35 year age range, self-reported being a current smoker, had a concussion in the past 30 days, were pregnant, currently taking medication to regulate mood, currently taking birth control medication, or taking marijuana or other memory-altering substances in the past 2 days. In addition, if they exercised 5 hours prior to the visit or consumed caffeine 3 hours prior, the visit was rescheduled. Lastly, participants were excluded if they answered "yes" to any of the 7 questions on the PAR-Q.

Exercise and Control Conditions

The control condition involved a time-matched (to the exercise condition) period of inactivity (sitting). To prevent boredom, they engaged in a seated task of playing an on-line puzzle. We have demonstrated that this control task does not prime or influence memory function (Bough & Loprinzi, 2019).

The participants were instructed to walk on a treadmill (Woodway treadmill) for 15-minutes. However, we aimed to have participants walk (moderate intensity) at 50%-59% of their estimated heart rate reserve (HRR), sing the following formula:

Target Exercise Heart Rate (bpm) = $[(HR_{max} – HR_{rest}) \times \%\ intensity] + HR_{rest}$

For the calculation of HRR, HR_{max} was estimated using the 220-age equation. For HR_{rest}, participants rested for 5-minutes before their HR was measured (via Polar HR monitor). Heart rate (Polar) was monitored throughout the exercise (Garber, Blissmer, Deschenes, et al., 2011).

Memory Task

The memory task was the Logical Memory Test, which has demonstrated evidence of convergent validity with the WMS-IV (Wechsler Memory Scale) logic memory test (Schnabel, 2012). It consisted of two short narrative stories that are 25 lines long. Both stories had sequential plots with congruent content and similar in length. There were feature phrases used to engage the participant's emotions. One memory assessment occurred immediately after the memory task, and the other was 25 minutes after the first assessment at the end of the visit.

For both stories (A and B), participants listened to the story via headphones. They listened to Story A once, followed by listening to Story B twice. Participants were told to

commit these stories to memory as they would subsequently be asked to recall as much information as possible from these stories. While some words are required to receive credit, other lines only require a variation of the phrase or words. Scoring is added up following the participant's recall of the story, and the higher the score, the better the memory performance of the participant. The maximum possible score for each individual story is 25 points. Further details on the scoring of this memory task can be found elsewhere (Schnabel, 2012). Memory Performance is shown in Table 5.

Social Stress Test

The Trier Social Stress Test was used as a model, participants completed 2 stress-inducing tasks (Birkett, 2011). We recently validated this psychological stressor in a young adult sample (ie, demonstrated increased cortisol levels from this exact psychological stress protocol) (Ponce & Loprinzi, 2019). Participants were first told to sit quietly and prepare for a speech (ie, being interviewed for their dream job) that they would be delivering to the research team. They were told that this speech would be recorded and shown to University faculty for review of public speaking strengths and weaknesses (this never actually happened). Additionally, they were told that they could use their notes during their presentation, but then right before the presentation, the notes were taken away from the participant. After 10-minutes of preparation, they delivered this audio- and video-recorded speech. Two researchers were always present during the 5-minute speech and 5-minute arithmetic task. After the 5-minute speech, participants completed a 5-minute arithmetic task that involved sequentially subtracting 17 from the number 2,023. See Table 1 for specific details on the employed social stress test.

Table 1

Instructions for the Social Stress Test

Speech Task	**Arithmetic Task**
"Please sit here and prepare for 10-minutes to give a 5-minute oral presentation. You should imagine you have been invited to your dream job interview, and you must convince the hiring committee that you are the best applicant for the position. Please note that you will be recorded by a camera and a microphone for subsequent voice and behavioral analysis. Members of the research team are trained in behavioral analysis and will take notes during your talk. You should try to leave the best possible impression. The researchers will reserve the right to ask follow-up questions in case of uncertainties to receive all necessary information from you. Following your talk, you will be given a second task, which will only be explained to you after your presentation. You may take some notes now, which you may use during your talk. The performance with the most mistakes will be shown to university faculty members, who will rank order the videos for use at a UM public speaking symposium assessing unsatisfactory public articulation skills of select university students. As you know, the University of Mississippi is committed to upholding the highest standards of excellence. Therefore, strengths and weaknesses of enrolled students must be properly analyzed to promote innovative curricular improvements." "Go ahead and start preparing for your presentation. I will come back after 10 minutes and we will get started." [10 minutes later]	[after the speech task] "During the next 5-minutes of this task you will be asked to sequentially subtract the number 17 from 2,023. You will verbally report your answer aloud and be asked to start over from 2,023 if a mistake is made. Your time begins now." If a participant makes a mistake, prompt them with "That is incorrect, please start over from 2,023." Set a digital time for 5 minutes.

"Please show me the notes you have taken in preparation. I know I told you that you could use these notes during your presentation, but per our protocol, you actually will not be able to use these notes. Please hand me your notes." "During your presentation, I will be writing notes. Remember, researchers may ask you questions about your speech." "Please stand over here while (researcher) starts the camera. Everything is ready, so please start your presentation. Your presentation will last for 5 minutes."	

Surveys

Before and after the social stress test, participants completed 2 quick assessments of affect and perceived arousal. The affect scale (known as the Feeling Scale) consisted of a one-item asking participants how they feel in the current moment, ranging from -5 (very bad) to +5 (very good), with 0 being the neutral point (Hardy & Rejeski, 1989). Additionally, at these time points, participants also completed the Felt Arousal Scale, which consists of a single item asking how physiologically aroused (ie, worked-up) they feel in the current moment, ranging from 1 (low arousal) to 6 (high arousal) (Svebak & Murgatroyd, 1985).

Additional Measurements

Body mass index (BMI; kg/m^2) was calculated from measured height and weight in the laboratory, using a SECA stadiometer and scale (Loprinzi et al., 2019).

Statistical Analysis

All statistical analyses were computed in SPSS (v. 24). A 2 (exercise vs. control) x 2 (immediate vs. delayed memory) repeated measures ANOVA was employed. Partial eta-squared (η^2_p) effect size estimates were calculated. Statistical significance was established as an alpha of 0.05 (Loprinzi et al., 2019).

Results

Table 2 shows the characteristics of the sample. Demographic characteristics (age, sex, race-ethnicity, measured body mass index) were similar for the exercise and control groups.

Table 2

Characteristics of the Sample

Variable	**Exercise**	**Control**
N	20	20
Age, mean years	20.7 (0.9)	20.5 (1.3)
Sex, % Male	35.0	40.0
Race		
% non-Hispanic white	65.0	65.0
% non-Hispanic black	15.0	30.0
% other	20.0	5.0
Body mass index, mean kg/m^2	26.9 (5.6)	25.5 (4.5)
Perceived Stress Scale, sum	-	-

Note.
Variance estimates in parentheses are standard deviations
-, not assessed

The exercise responses for heart rate can be found in Table 3. The mean heart rate at the end of the exercise bout was 147.3 bpm.

Table 3

Exercise Responses

Variable	**Exercise**	**Control**
Resting HR, mean bpm	84.3 (16.5)	86.2 (13.9)
Mid-Point HR, mean bpm	139.7 (17.1)	80.7 (11.1)
End-Point HR, mean bpm	147.3 (16.2)	77.3 (12.2)
End Exercise RPE, mean	-	-
2-min Post HR, mean bpm	97.8 (16.9)	80.2 (11.3)

Note.
BPM, beats per minute
HR, heart rate
RPE, rating of perceived exercise
Variance estimates in parentheses are standard deviations
-, not assessed

Table 4 displays the social stress responses. After the social stress test, heart rate was elevated by approximately 10-20 bpm. The exercise and control groups, affective response (Feeling Scale) were drastically reduced, indicating they felt worse after the stress test. The exercise and control group, perceived physiological arousal levels increased after the social stress test. The mean pre- and post-stress appraisal levels, for both groups, were close to 3 (range 1-5). This indicates that, on average, participants did not strongly agree or strongly disagree, but rather, neutrally-agreed, that the stressor was a negative experience. That is, they did not agree or disagree that the stressor was a negative experience.

Table 4

Social Stress Responses

Variable	**Exercise**	**Control**
Resting HR, mean bpm	84.3 (16.5)	86.2 (13.9)
Baseline Affect, mean	2.8 (1.3)	2.4 (1.7)
Post Social Stress Test Affect, mean	1.6 (2.0)	0.7 (2.6)

Baseline Arousal, mean	2.3 (0.9)	2.1 (1.1)
Post Social Stress Test Arousal, mean	3.6 (1.0)	3.5 (1.2)

Note.
BPM, beats per minute
HR, heart rate
Variance estimates in parentheses are standard deviations

Table 5 shows the memory performance scores. There were no statistically significant main effects for group or group x time interaction, but there were statistically significant main effects for time.

Table 5

Memory Performances

Variable	**Exercise**	**Control**	**Test-Statistic**
Pre Story A, mean	9.9 (3.6)	11.1 (4.0)	F(time)=21.0; $p < .001$; $\eta^2_p=.36$
Post Story A, mean	8.8 (4.1)	9.1 (4.1)	F(time x group)=1.77; $p = .19$; $\eta^2_p=.04$
Pre Story B, mean	15.5 (3.7)	16.9 (2.9)	F(time)=27.46; $p < .001$; $\eta^2_p=.42$
Post Story B, mean	14.3 (4.5)	14.7 (3.0)	F(time x group)=2.24; $p = .14$; $\eta^2_p=.06$

Note.
Variance estimates in parentheses are standard deviation

Discussion

Previous work indicates that psychological stress induction before memory encoding and during memory consolidation can impair subsequent memory retrieval (Gagnon & Wagner, 2016). Further, acute aerobic exercise prior to memory encoding and during memory consolidation has been shown to enhance memory retrieval (Loprinzi,

Edwards, & Frith, 2017). Memory recall declined across the 2 time points (significant main effect for time), however, this decline was similar between the exercise and control group (no significant group x time interaction effect).

We anticipated that acute aerobic exercise prior to memory encoding and during memory consolidation, when compared to no exercise, would associate with memory function even when being exposed to a stressful environment. This hypothesis is grounded in previous work showing that 1) acute exercise during both of these time periods can enhance memory function, including stress-related implicit memory (particularly in animal models; these studies, however, focus on exercise and its effects on stress memories [fear conditioning]) (Loprinzi & Edwards, 2018) 2) exercise *prior to* a stressor can facilitate emotional regulation after the stressful experience (Edwawrds, Rhodes, & Loprinzi, 2017), which may help subserve memory function (Pascuzzi, 2017), and 3) relatedly, exercise may help to facilitate emotional resilience to an acute stressor (Childs & de Wit, 2014).

Our inability to demonstrate an effect of acute aerobic exercise on memory function before being exposed to a stressful environment may be a result of several factors. First, despite the plausibility of our hypotheses, it is possible that acute aerobic exercise does not play a causal role in improving memory function after exposure to a stressful stimulus. Future research will be needed to confirm or refute this. Notably, emerging work in mice indicates that exercise that co-occurs with stress can help mitigate memory impairment and facilitate cellular mechanisms of memory (Miller, Marriott, Trotter, et al., 2018). However, if exercise does play a causal role in humans, then there are several potential explanations for our null findings. Second, despite previous work

demonstrating that acute stress close to the retrieval period may impair memory retrieval, additional work should carefully consider the temporal effects of the stressor (Park, Zoladz, Conrad, et al., 2008; Guez, Saar-Ashkenazy, Keha, et al., 2016) . It is plausible that if a stressor occurs earlier on in the protocol, either before or immediately after memory encoding, this may result in a greater stress-related memory decline, and possibly, allow for a greater ability for acute aerobic exercise to attenuate this effect. However, this is unlikely, as the experiment included the acute imposed aerobic exercise and psychological stress prior to memory encoding. Third, we may have observed null findings due to the brief consolidation period selected for this experiment, which could have prevented stabilization of the weak memory trace, or perhaps the cognitive demands associated with degree of difficulty, and/or lack of personal saliency to allocate cognitive resources to the effortful storage and recall of the Logical Memory task. Following encoding, a variety of neural mechanisms fortify labile memories within a time-dependent consolidation interval (Roig, Nordbrandt, Geersten, et al., 2013; McGaugh, 2000). Therefore, recall assessments executed too soon after initial encoding risk futility if the consolidation process is still underway. In addition, Roig et al cautions against assessing memory too soon following the exercise stimulus, as more robust effects on long-term recall may emerge at, or exceeding, about 24 hours post-exercise (Roig, Skriver, Lundbye-Jensen, et al., 2012). Fourth, perhaps exercising prior to stress induction was sufficient to benefit emotional regulation in response to acute social stress, but not to a degree that would meaningfully facilitate recall performance for this sample. Future work should investigate whether other exercise intensities or modalities may induce an enduring impact on emotional reactivity, capable of affecting critical memory

outcomes. Relatedly, it is plausible that acute exercise, based on the affective response, may either exacerbate or mitigate a stress-induced memory impairment effect. As such, future work should evaluate the potential extent to which acute exercise-induced affective response mediates this paradigm, as well as how habitual engagement in exercise moderates these potential effects.

It is possible that our stress protocol was not sufficient to elicit a high stress response. Despite our findings that our stress protocol worsened affect (feeling scale) and increased perceived physiological changes (felt arousal scale; Table 3), these responses did not produce an extreme effect on the evaluated scales. For example, affect was reduced from approximately 3 to 1 after the stress protocol (Table 3). This affect scale ranges from +5 to -5; thus, demonstrating that the stress protocol did not induce a perceived stress response that reached an extreme level. Of course, if participants viewed these extremes as the most/least stressed they have ever been, then an extreme rating would have been considered unlikely or perhaps inappropriate. Further, experimentally inducing extreme stressors may pose a substantial risk to participants' psychological well-being during the experiment and after leaving the laboratory, which may be unethical. Another possibility is that extraneous stressors, unrelated to our protocol may have influenced participants' change in stress. That is, if college participants were already moderately stressed, even a well-established social stress assessment may not have been potent enough to elicit a statistically meaningful decrement in global affect. Finally, although our administration of the Trier Social Stress Test was similar to standard methodological procedures, due to feasibility concerns and participant time-burden, we did not confirm that participants had engaged in 30-45 minutes of rest to offset

detrimental effects of prior stress upon entering the laboratory (Kudielka, Hellhammer, & Kirshbaum, 2007). Additionally, the primary researcher and confederate were not dressed in white lab-coats, and only one research confederate was utilized in addition to the primary investigator (rather than the 3 to 4 minimum team members recommended), which may have lowered the physiological ceiling of the anticipated stress response (Kudielka, et al., 2007). However, despite these possibilities, our stress protocol did worsen affect and increased perceived arousal.

These affect and perceived arousal levels align with the pre- and post-stress appraisal responses, which, similarly, had mean values (neutral) in the middle (Table 3). However, this stress protocol has previously been shown to impair episodic memory (Guez, et al., 2016). Third, despite our self-selected walking protocol being sufficient to enhance memory function, it is possible that our exercise stimulus did not occur at the ideal intensity level (Sng, Frith, & Loprinzi, 2018). This aligns with our previous work demonstrating that high-intensity exercise has a protective effect in facilitating emotional regulation (Edwards, et al., 2017). However, a high-intensity bout of exercise may induce a large increase in cortisol, which may have a negative effect on memory, as the cortisol-memory relationship may fall along an inverted U-shaped relationship (Gagnon, et al., 2016). This, however, has been questioned by others (Chen, Nakagawa, An, et al., 2017). Notably, the cortisol-memory relationship is very complex, which is influenced by a multitude of parameters, such as the type of stress, duration of stress and content of the memory stimuli (Gagnon, et al., 2016). Further, subsequent work should examine whether high-intensity exercise preserves or further depletes the memory trace, despite heightened cortisol secretion.

Limitations of this experiment include the utilization of a non-population-based representative sample (college students), the relatively small sample size per group, only recording exercise-induced physiological responses (heart rate) at the mid- and end-point of exercise (as opposed to throughout the entire bout of exercise), and implementing an exercise intensity with participant heterogeneity. In regards to this, the relative exercise intensity was 74%. Regardless, future work should more carefully elicit the desired relative exercise intensity, with minimal variability across participants. Another potential limitation is that we did not employ a non-stress control group (ie, a group without the social stress protocol). Although this would potentially have been useful, it was not central to our research question, which was to see if exercise (vs. no exercise) is associated with memory function when being exposed to a stressful environment, as opposed to seeing if stress (vs. no stress) was related to memory function, which has already been demonstrated. Clearly, there is considerable work on this topic that is needed, including addressing these study limitations as well as implementing our suggestions noted in the previous paragraph. Future work would also benefit by addressing this paradigm but extending the follow-up memory assessment (ie, assess long-term memory). Additionally, future work should consider evaluating if individual differences in pre-experimental perceived stress may drive psychological vulnerability to social stress induction, as well as the potential protective effects of exercise on attenuating stress-related memory decline. Despite these limitations and need for future work, strengths of this study include the experimental design, addressing a novel paradigm, and incorporating both subjective and objective measures of stress.

In conclusion, this experiment demonstrated that acute aerobic exercise (including self-paced and imposed exercise), occurring prior to memory encoding or during memory consolidation, was not associated with memory function after exposure to a stressful environment. Future work in this under-investigated line of inquiry is needed.

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